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Some common heat manifestations and hyperpyrexial conditions

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SOME COMMON HEAT MANIFESTATIONS

AND

HYPERPYREXIAL CONDITIONS

By

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SENIOR THESIS

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INTRODUCTION

Heat manifestations are not new nor have they been described only in recent times. It has been known for more than 2000 years that a high external temperature may exert a deleterious effect upon the human body. In Jonah IV:8 there is reference to a probable victim of heat stroke.

"And it came to pass, when the sun did arise, that God prepared a vehement east wind; and the sun beat upon the head of Jonah, that he fainted, and wished in himself to die, and said 'It is better for me to die than live.'" In Isaiah XLIX:10 and Psalms XXI:6 there is a caution against the danger of being smitten by the sun. Again in 2 Kings IV is the case of the Shunammite woman's son. The Arabians called the symptoms due to excessive heat, Siriasis, after Sirius the Dog Star.

Corden in the sixteenth century recognized the disease and thought it was apoplexy due to heat --- morbus atlonitus. It was regarded in the eighteenth century by Baerhaaue as phrenitis. It wasn't until the nineteenth century, however, that the Anglo-Indian Surgeons and the physicians of the United

States gave us a full knowledge of the different affectations due to excessive heat.

Before presenting the disturbances or derangements, it is best to present briefly the normal physiology of the mechanisms involved.

The mechanisms which regulate heat dissipation (caliber of skin vessels, sweat glands, respirations) are doubtlessly largely controlled by nerves; and, hence, ultimately involves the vasomotor, sweat, respiratory and cardiac centers in the medulla; although an array of evidence can be assembled also in favor of strictly spinal reflexes.

It is known that activation of the vasoconstrictor center such as postulated in reaction to cold produces predominant constriction of the splanchnic vessels; whereas, in cold reactions the constriction is rather explicitly limited to the skin vessels, while those, of muscular and visceral portions dilate. The involvement of the higher centers is demonstrated by the fact that the regulation of heat dissipation is not abolished by progressively higher sections of the brain-stem until the cut is made in the diencephalic region. The preponderance of evidence strangely suggests that still higher centers exist which act to coordinate the heat

producing and heat dissipating centers of the hypothalamus.

There are two mechanisms or theories concerning the manner in which it is set into action:

- (1) The center or centers may be stimulated by alterations in temperature of the blood, or
- (2) They may be controlled reflexly.

The heat centers represents a series of nuclei upon which many impulses impinge. Afferent impulses causing inhabitation of the mechanism tending to increase temperature, arrive not only from the heat receptors of the skin but from muscles and viscera as well. Since the number of inhibitory impulses exceeds the excitatory, the center is set at a thermostatic level regarded as normal temperature. When either the excitatory impulses are increased or the inhibitory decreased the center is reset, so to speak, as a thermostat operating at a higher level.

Heat is normally lost in various ways: About 5% by warming of food and respired air, and with the excretions; another 15% by saturation of air in the lungs with water, and by heat absorbed in eliminating CO_2 from the lungs. The remainder (80%) is lost by radiation, conduction and water vaporization from the

skin. The amount of water vaporized is determined by content in the skin, by the degree of sweating and by the temperature, humidity and movement of air in accordance with physical principles.

Temperature is influenced also by taking foods, hot or cold environments, and particularly by muscular work. The various heat manifestations have been known as:

Heat exhaustion, heat stroke, sunstroke, heat cramps, miner's cramps, fireman's cramps, etc. Many of these conditions have derived their various names from the occupations in which the men are engaged.

DEFINITIONS

Heat exhaustion is a condition closely resembling shock, characterized by sudden onset; with weakness, dizziness, nausea, uncertain gait, pallor, and then temperature is usually subnormal.

Heat stroke is a condition characterized by sudden onset, sudden high temperature, and loss of consciousness. There is usually mental excitement and delirium early, proceeding to hyperpyrexial coma, stertorous breathing, face flushed and cyanotic, pupils early dilated

and contracted in comatose stage, fibrillary twitchings of muscles and convulsions common. Incontinence of feces and urine is frequently found.

Heat cramps (Fireman's cramps, minor's cramps, etc.) is a condition characterized by pain in various groups of muscles which prevents the subject from continuing work. There may be nausea and vomiting, and there may or may not be an increase in body temperature.

ETIOLOGY

The common etiological factor present in the various manifestations is heat.

There are many ways in which the factor makes itself present. The most common and perhaps the best known is direct exposure to the rays of the sun. The various component rays of the sun have been studied individually in an attempt to prove or disprove their influence on the production of heat conditions. The relative humidity under various temperatures also plays an important part. Some of the other factors which have been noted to influence the patient's reaction to heat are: occupation, physical condition, alcoholism, clothing, race, age, and sex. And it is the various

combinations of these factors that tend to determine in some measure the type of clinical disease with which the patient is stricken.

One of the earliest references in American literature in regard to heat effects, is an article taken from the Medical and Surgical Reporter for 1878 (48).

"A writer in the 'Enterprise' of Virginia City, Nevada, gives a striking description of the effects of the great heat in the mines on men exposed to its action. The temperature in the shafts is from 120° to 130°F. The writer says that this temperature, so far above the natural heat of the body, attacks it and causes the flesh to undergo the first stages of cooking. This happens to a miner the moment perspiration ceases to flow from the pores of his skin. The stomach is first affected, then the brain. As soon as this occurs the body begins to cook, and first of all, apparently, the brain, as the man at once becomes delirious---"

It is interesting to note that at this date a rather clear concept of the order of events and pathology present was known.

To contrast with this idea is the stand taken by Sambon (53) in an article written by him in 1898. He calls it Siriasis, after the Ancient manner of

naming it from Sirius the Dog Star, not heat fever, but an infectious disease.

Samson in his study of the disease found the following ideas concerning its etiology: " --- a gradual heating of the blood to a degree of incompatibility with the maintenance of nervous function --- heat paralyzes the centre or centres which are supposed to regulate the disposition of the bodily heat, and thus cause the hyperpyrexia and other symptoms of the disease."

He found that still others believed that the resulting paralysis caused a great heat production and still others thought that it caused retention of heat. In some of Little's works that Samson read, he found that Little considered it to be the result of pressure exerted upon the cerebrospinal matter by the heat expanded cerebrospinal fluid. Some of the others were Antavini's Acute Neurosis of the vaso-motor system, Laveran's paralysis of the ganglia of the heart, Valliu's coagulation of the myosin, and Baxter and Zuber's interference with heat loss as a result of suppression of cutaneous perspiration.

Because of this wide diversity of opinions, Samson came to the following conclusions: "The symptoms

of the disease, its relapses, its morbid anatomy, its peculiar geographical distribution, its epidemic outbursts, the relative immunity to its attacks afforded by acclimatization all clearly point to the specific infectious nature of the disease."

HEAT EXHAUSTION

Of the three main types of heat affections heat exhaustion is the least serious of the three.

Heat exhaustion is generally conceded to be vasomotor palsy and has as its main features lowered temperature and a shock-like condition. It is a manifestation in which the existence of heat paralyzes the center or centers in the medulla and the bodily heat is dissipated much more rapidly than it is produced (56).

Heat exhaustion therefore presents a feeble weakened condition or a state of collapse with a lowered body temperature which may range from below normal to 94° F. As a general rule it occurs in old people or in those individuals whose body resistance has been lowered (56).

The patient is usually brought in appearing flushed to cyanotic, the skin is damp and cold, the

respiratory movements are quite rapid and shallow, the pulse is rapid and thready, and the temperature is normal to subnormal. The mental state of the patient varies from slight confusion to semicomatose states. In spite of the doubtful appearance that patients sometimes make, the prognosis on a whole is usually very good. These patients then with adequate and prompt treatment make a rather uneventful and uncomplicated recovery. It is true, however, as it is in other conditions that pre-existing organic pathology may make the treatment of these cases a definite problem and the prognosis exceedingly grave.

The usual line of treatment in these cases is first to remove the patient to a cooler atmosphere. There loosen any tight clothing or undress them and place them in a hospital bed.

The heat loss should be combatted by the application of heat and extra blankets. Hot stimulating drinks such as coffee or tea given. These can be given orally if the patient is conscious or per-rectum if he is unconscious. When the initial symptoms have passed, the treatment evolves itself into symptomatic care.

Because of the relatively mild and uncomplicated nature of heat exhaustion as a whole, I wish to take

the liberty to conclude further discussion on heat exhaustion and limit the rest of my paper to heat cramps and heat stroke.

HEAT CRAMPS

Attention was brought to heat cramps because of the common prevalence among industrial workers and stokers aboard ships.

In heat cramps there may be a prodromal period of 1-3 days before the onset of any acute symptoms. One of the symptoms may be diarrhea (61).

Pain in various groups of muscles which prevents the subject from continuing work is one of the presenting symptoms, also tingling of the fingers and toes may proceed or accompany the cramps (61).

It has also been noted that there is a predilection of certain groups of smooth and striated muscles involved in the cramps.

The most common groups are found to be the calf muscles and those of the flexor group of the forearm. Then too the muscles of the abdominal wall are found to be effected. On some instances quite apart from the involvement of the abdominal muscles the smooth

muscles of the gut were effected. This is believed to possibly be the cause of the vomiting and diarrhea. There being set up an irritation similar to that produced in the skeletal musculature (61).

Willcox (66) advances the opinion that there is not a common causal agent in both heat cramps and heat strokes. He noted that the convulsions in heatstroke were made worse after intravenous saline injections. Thus the opposite effect might well be expected if the essential pathological process were a depletion of salt and water in the central nervous system.

One important factor in substantiating the diagnosis of heat cramps is the great relief following intravenous injection of large amounts of normal salt solution.

Some of the etiological factors in the production of heat cramps are:

1. Exposure to a high temperature at work.
2. Rapid loss of salt in the sweat that is not replaced.
3. Diminished concentration of chloride and base in the blood and likewise in the body tissue.

PATHOGENESIS

Several theories have been advanced as to the explanation of the cause of heat cramps.

Edsall (18) in 1904 described two cases with muscle spasms which occurred after exposure to severe heat.

"One of the most striking features was a very conspicuous degree of fibrillary contraction, particularly in the muscles of the calf. This was of continuous occurrence except during the frequent interruptions caused by the most marked of all three symptoms, namely, severe tonic spasms." He then goes on to state "That the spasms usually lasted about 30 seconds to a minute and that when they occurred they were exceedingly painful."

He states that the muscles usually involved were those of the forearms and legs, also the muscles of the abdomen were sometimes acutely involved. In their gross features the tonic spasms very closely resembled tetany, with the exception of the pronounced abdominal involvement. He tested the patients for Chvostik's, Trousseau's and Hoffmann's phenomena but these were absent. The most striking and characteristic

feature of all was the extreme irritability of the muscles to direct stimulation. The spasms occurred spontaneously quite frequently, but any attempt at voluntary use brought about the spasms. The spontaneous spasms were nearly all gone within a twenty-four hour period. There remained, however, for several days an irritability to stimuli, and a very abnormal degree of fibrillary response would result. These factors lead Edsall to propose that acute degeneration of the muscles to be the cause of the heat cramps (18) (19).

Haldane in 1923 (33) (34) advanced the idea of kidney disfunction due to the excessive demands made upon it by the heat and the accompanying factor of a type of secondary anemia. The kidney function being impaired, also the accumulation of water in the tissues, as a result of this a water poisoning takes place and the musculature goes into spasms.

M. S. Elliot notes that Stokers on men-o-war have tonic spasms of the forearms, legs, thighs and abdominal muscles. The condition he associates with extreme excessive sweating. However, he made no comment beyond that as to the etiological agents (21).

In an article written by K. N. Moss in 1923 (42) are some interesting observations.

The observations were made upon Colliers. The men that were effected by cramps usually were so affected in the second half of their shift. The muscles involved were those that were being strained at that time. The men were usually those with poor physique. He also made note of the fact that urine examination showed small amounts of urine being voided with a relatively low chloride content. Moss also upholds Haldane's conception of water poisoning and quotes Pembrey as follows ---"Pembray showed that in hard work there is complete, or almost complete, cessation of kidney excretion, doubtlessly owing to the blood being diverted to the muscles."

It remained then for Talbott and Michelsen in 1933 (61) to bring to a clear conception of factors producing heat cramps and the part they played.

They found that the body tissues of patients with cramps contain hypotonic fluids, and this condition existed not because of the excess water, but rather because of the depletion of total base and chlorides. They likewise found that after the beginning of treatment that water and salt were retained to make up the deficit. This followed then that if this assumption were true, there would be a resulting gain in body

weight. But if the reverse were true, the cramps were on a basis of water poisoning and the fundamental process were one of increased total body water, then when treatment was started there should be a resultant loss of weight due to the diuretic action of salt and water. The facts remained that when treatment was started the patient gained weight.

They also found regarding the kidney function that the principal reason for low kidney function during the working hours in heated atmospheres was a peripheral dilatation of the capillaries for the dissipation of heat rather than the primary shut-down advocated by Haldane and Pembrey (61).

Talbott and Michelsen found that the reduction of blood chlorides ranged from 2%-10%, and while they had no direct evidence to show that the drop in blood chloride level is followed by a similar depletion in the chlorides of the intercellular spaces, it usually follows, however, and is generally assumed that the concentration of electrolytes in the blood serum and of the intercellular spaces are approximately equal. These facts then assumed to be true, it follows that the loss of water and salt from the blood serum is

partially replaced from the tissue, and there is a general hypotonic condition in both the blood serum and intercellular fluid (61).

It followed then that in these conditions there is a loss of salt and water from the body with a concomitant replacement of only the water which resulted, if this process were continued, by an eventual lowering of the chloride level below the normal range. It is their contention that when the critical level of the chlorides was reached in the working individual, muscle cramps occurred. What this critical level is depends upon the individual's susceptibility, acclimatization and the length of prodromal period (61).

Two years later Storkov and Tikesh (57) arrived at the same conclusions. They quote Weirich who states that the normal water-loss of man under normal conditions at rest is about 550 c.c. They found in their studies that during a working shift of 4 hours and 48 minutes that a loss as high as 10,000 c.c. was not uncommon. Some acclimatization seems to take place, because there would be a sweat loss of $1/2$ L. per hour in a new man compared to the $3/4$ - $1\ 1/2$ L. per hour loss of the men accustomed to the conditions.

In order to carefully study the clinical phases they hospitalized all the men, who worked in hot departments suffering from heat cramps and prostration. This was done for twenty days. Forty men were sent within one-half hour following onset of symptoms. Blood viscosity and hemoglobin content were taken immediately along with temperature and pulse rate. Each patient was weighed, and a sample of urine was taken to determine its chloride content. During the length of time the patients were in the hospital records were kept of: Food, water and salt intake, urine and feces output, and the weight was recorded daily. Division was made into two groups; those who had cramps and those not having cramps (57).

The following observations were noted: Severity of the disease was directly related to the blood and urine changes. Viscosity and hemoglobin content high at the onset gradually decreased reaching normal in four or five days. The body weight increased in the first twenty-four hours. This continued during the stay but gradually diminished until it became stable (57).

The chloride content of the urine was characteristic in that it was very low in the beginning, and gradually increased to normal by the third hospital day. Also

the output varied with the severity of the case, being very low on the first day (57).

They found on the other hand that in heat exhaustion these changes were very slight, and depended upon the length of exposure and disturbance in water-salt metabolism. Then too the heat exhaustion cases occurred chiefly among the new men, and was probably on a basis of heart weakness and inadequate thermoregulation. The sweat glands not functioning as generously as they seemed to maintain the proper heat balance. The evidence for this lay in the high urinary output of these patients with a urinary chloride content within normal limits (57).

They thus concluded that heat cramps and exhaustion were separate diseases. In cramps, the etiological factor was water-salt metabolism disturbance; whereas, in heat exhaustion, the cause is in the disturbance of thermoregulatory system.

SALT METABOLISM

The daily intake of salt and the ordinary urinary output in man nearly parallel, and is about 10-15 grams. However, there is opened another avenue of elimination

when the sweat glands become active. The glands secrete a hypotonic solution containing sodium and potassium chlorides. Also there is low concentrations of lactic acid, urea and other substances. The composition of sweat is not stable or uniform. It is dependent rather upon the conditions existing in the environment both internal and external (16).

The mechanism for controlling this salt concentration remains at the best obscure. It has been suggested that the skin acts as a depot for the salt. Thus perhaps the products of the sweat glands activity are dependent upon the concentration in the skin. Also it is possible that the saturated salt solutions which are present on the skin during sweating in dry heat exert enough back pressure on the sweat glands so as to modify their activity.

Thus there is maintained during profuse and continued sweating high concentrations on the skin (16).

As the one proved function of sweating in normal man is the part it plays in temperature regulation, it would be no disadvantage to him if sweat were free of the dissolved electrolytes, because the kidneys and lungs are quite normally able to take care of the maintenance of acid base equilibrium. Thus it can

readily be seen that the sweat glands can easily upset this equilibrium disastrously, and heat cramps insue from the loss of salt (16).

CIRCULATORY EFFECTS

It is interesting to note the effects of heat upon the circulating blood. Sweating and dilatation of the skin capillaries is brought about by conditions of extreme exposure, and with such conditions present, CO₂ may escape by means of the sweat and most likely to some extent through the layers of the skin. It has been attained by the calculation of data of various workers that the CO₂ loss through the skin is exceedingly large. It was worked out also that the sweat is always more acid then the blood, even after prolonged exposure. Thus all the losses through the skin result in fairly large balance of alkali remaining in the blood stream. In man the rapid breathing may be an answer to the demand for cooling. The process is not entirely involuntary, and so when it does occur it may lead to over ventilation (1).

Adolph and Fulton also found some interesting facts in regard to peripheral circulation and blood

pressure. They found that the diastolic blood pressure fell while the systolic pressure quite frequently was elevated 20 mm. of mercury. The systolic pressure, therefore, was acting to compensate for the fall in the diastolic pressure. There is also present an increase in pulse rate accompanying the systolic rise in an effort to compensate for decreased peripheral resistance. It was found too that the venous blood was much redder than normal, also indicating a more rapid circulation through the body (1).

They explain the drop in diastolic pressure as being the result of the relaxed condition of the peripheral vessels. This relaxed condition of the vessels then would account for the flushed appearance of the individual (1).

In exposure the response of the capillaries is mainly as follows: To begin with the capillaries expand being stimulated by the warm temperature sense of the skin; and consequently radiate heat from the skin. However, because of the surrounding increased temperature and humidity, the radiation results from such a process is practically nil (1).

Now that the circulation has started to fail there is added to the picture a new factor namely the one of

chemical control of the circulation in the capillaries. It is believed that the pituitary secretion acts as a constant excitant to the capillary bed to thus maintain them in a contracted condition. Whenever the circulation seems to slow or stop, there is then sent a new supply of the chemical substances. This supply fails to reach the capillaries because of the inadequacy and subsequently the tone of the capillaries is greatly decreased until the expansion of these vessels is extreme (1).

The venous filling of the heart to some extent controls the tachycardia. Therefore, the heart sometimes compensates for the lack of venous volume by increasing in its rate. Thus from these facts can be involve the following theory:

External stimulation of the skin leads to a reflex sweating with often a progressive deeping and acceleration of the respiratory movements. Thus CO₂ is lost from the blood vessels in process of sweating. The resistance of the peripheral circulation is greatly reduced due to the expanding of the cutaneous vessels. As sweating progresses the volume of the circulating blood may be quite significantly reduced. The heart in turn compensates for the reduced venous volume by

gradually increasing its rate. There is thus produced in exposure to high temperatures a type of circulatory failure which is in many ways comparable to the general opinion of shock (1).

Thus Adolph and Fulton drew the following conclusions:

"1. Exposure to high temperatures increased the loss of carbon dioxide from the blood through the skin and lungs. This lowering of the carbon dioxide tension increased the hydrogen ion concentration of the blood and ultimately leads to an excretion of alkali from the blood.

"2. The peripheal blood vessels are greatly dilated during exposure to high temperatures and this dilatation continues indefinitely. The lack of a high resistance in the peripheral blood vessels prevents blood from returning to the heart.

"3. The heart rate increases steadily and rapidly and is even able to increase the systolic blood pressure. In spite of this compensating activity on the part of the heart, the blood flow back to the heart finally becomes inadequate. At this point circulatory failure or shock is complete with faintness.

"4. The rise in skin temperature seems to play

the initiatory part in the control of the respiratory and circulatory reactions." (1)

It has been quite closely pointed out by the work of the above men that while heat cramps is on the surface quite simple, the effect on the human organism is fairly wide spread and that a reasonable working knowledge of the functions of the systems involved must be had.

Also consistant with the ideas of effect upon the working man of high temperatures is the work of Vernon and Warner (64). They studied the effect of humidity at high temperatures upon the capacity for work. The pulse beat usually was about ten beats greater in **hot** dry air than in the moist air of the same wet-bulb temperature. The pulse rate and temperature correspond because it was higher (0.3° - 0.6° F.) in the dry air than the moist wet-bulb temperature. Above 70° - 75° F. the gross mechanical efficiency fell off slightly. Also the weight of the moisture which was lost by sweating corresponded well with the effective temperature scale. It was increased gradually in dry air and somewhat diminished in those made in moist air (64).

The degree of fatigue experienced in dry air was considerably greater than in moist air of the same

wet-bulb temperature (64).

PROGNOSIS

The prognosis is on the whole quite favorable. The men seemed to have no resultant permanent disability when they had recovered from the attack. The convalescence of the attacks of heat cramps is usually from four days to a week. At the end of that time they seem quite able to return to their former work.

As a rule there does not seem to be any acquired weakness which may predispose to further attacks if the patient takes pains to carry out simple routine prophylactic measures.

TREATMENT

It can readily be seen from the nature of the disorder what the therapeutic measures are.

First, prompt first aid should be administered; which is essentially removal to a cooler atmosphere,

Second, the restoration of normal body temperature should be strived for.

Large quantities of intravenous saline should be given. The amounts varying with individual needs. Also

normal saline with 5-10% glucose is extremely useful in these conditions.

Schofield also advocates sodium chloride tablets grains XV in four ounces of water every four hours.

Glover (30) found that the men who were using salt tablets or salt water had as a whole fewer cramps. He found that a sodium chloride table of grains XVI, one tablet taken every time the individual drank water, greatly lowered the incidence of cramps. These tablets were placed in inexpensive dispensers above the drinking fountains, and the men instructed in their use. In some instances the water which they drank had in it small amounts of sodium chloride. It was found, however, that the men objected to the taste and preferred the salt tablets.

HEATSTROKE

Heatstroke, hyperpyrexia, or thermic fever is the most serious of the heat manifestations and is the most difficult to manage.

The etiology of the disease is exposure to extreme high temperatures. It may be the culmination of several hot days or the prolonged exposure to excessive heat.

It is more commonly found in laboring men and usually in the middle decade of life. Disease and alcoholism have bearing also on the susceptibility. Food, clothing and humidity have also been advanced as factors having bearing on the susceptibility. The individual susceptibility is a factor that must not be lost sight of, even though it can not be fully evaluated.

Because of the importance that some of the etiological factors play in the production of hyperpyrexia it would be wise to give them some consideration.

TEMPERATURE

"In January 1774 and April 1775 Dr. George Fordyce and Dr. Blagden exposed themselves to dry and moist heat in a specially prepared room. They found that they could remain in a hot dry room at 260° F. with apparently no discomfort or rise of body temperature for 13 minutes, while a beefsteak was well cooked (assisted, however, by blowing the hot air on it with a bellows) and eggs were roasted hard. Exposure to a moist atmosphere at 130° F. for 15 minutes caused a rise in mouth and urine temperature to 100° F. The value of the above experiment is to be doubted, but it does give some idea as to the

fact that the body may stand great extremes of temperature for short periods of time only. It would seem then, as Hill (38) brought out in 1920 that, "Heat stroke results from the rise in body temperature to height incompatible with the maintenance of the equilibrium of the physico-chemical reactions in the cell on which life depends."

Such rise in temperature too may result from an inability of the heat-regulating mechanism to control the body temperature under the existing atmospheric conditions. It also may be due to exhaustion of the mechanism from the effect of infection, drink, fatigue, which by weakening the mechanism embrace the risk of heat stroke.

Willcox was of the opinion that a maximum shade temperature of 110° F. appeared to be the dangerous level. Also a succession of several hot days was dangerous. This cumulative effect might not make its appearance until in the night or early morning when the atmospheric temperature had fallen considerably and the cause had apparently been removed (66).

HUMIDITY

This is an important factor in heat stroke. Because of the already saturated condition of the air, any attempt to further saturate it by evaporation from the body surface is nullified. The effect is further increased when the air is stagnant.

Hill (37) found that when surrounded by stagnant moist air at body temperature, any loss of heat by convection, radiation or evaporation of sweat becomes impossible, and as the heat production continues the body temperature rises. This rise in temperature in turn accelerates the rate of combustion in the cells, and the result is a vicious cycle being established.

Normally, he found, that in comfortable cool surroundings the individual is cooled mainly by convection. The cool moving air carries away the body heat from the skin.

At this time loss by evaporation is kept at a minimum, while the evaporation loss from the mucous membranes of the respiratory system is kept at a maximum, by the breathing of cool air at a low vapor tension. The skin vessels are toned up and activity and metabolism kept at a good level. In warm stagnant

air, however, the reverse is true and the emergency method of cooling is brought into play (38).

Wind, too, enormously increases the evaporation power as long as the air is saturated at a temperature below body temperature. Thus continuously unsaturated layers of air are brought in constant contact with the body (38).

Every gram of water which is evaporated either from the skin or respiratory membranes carries away 0.59 calories. Therefore, the cooling effect of the emergency system can be seen. When men do hard physical labor this is brought into play. It has been found also by Hill that the limits of power to keep cool is surpassed when the wet-bulb temperature exceeds 88° F. (in still air) even when the individual be stripped to the waist and resting (38).

In warm dry air at temperatures near the body heat. There is no danger as long as the sweating mechanism functions and the air remains in motion. However, on the other hand, in hot dry air well above body temperature, and with a wind, the cooling effect due to evaporation of sweat may be quite surpassed due to the heating power of the wind. The hot dry wind dries the skin, and the absorption of the sun's rays

takes place, which is normally checked by a protective layer of sweat (38).

The normal heat loss of a resting man is about one millicalories per square centimeter of body surface per second. Work may raise this to three millicalories. Clothing in exposure to the sun absorbs about 5-10 millicalories per square centimeter per second. Thus the strain on the sweating mechanism is greatly increased (38).

SWEATING

The suppression of sweating undoubtedly has great effect upon the mechanism of production of heat stroke. Hearne found that with the suppression of sweating the body temperature tended to adjust itself, in accordance with physical laws, to the temperature of the atmosphere which it may be in at that time. He also believed that cessation of perspiration was probably due to the exhaustion of the sweating mechanism having been under a strain produced by several days of excessive high temperatures (35).

It has also been suggested that the suppression of sweat is due to a paralysis of the secretory

mechanism of the glands produced by circulating toxins which are some unusual products of metabolism.

The other factors such as infection, alcoholism, fatigue, work in that they produce a general lowered resistance to any sudden or extensive demands made upon the body.

SYMPTOMS

The symptoms of heat stroke usually appear in the following order: There is a rapid onset of sudden high temperature. There is present malaise, nausea and vomiting. Frequently headache and restlessness occur. Frequent micturation may be a characteristic early symptom accompanied at times by urethral pain. There then follows mental excitement and delirium with hyperpyrexial coma, stertorous breathing, flushing of the face, and cyanosis and conjunctival congestion. The pupils at first dilate, then later contraction in the comatose stage. It is common to find fibrillary twitching of the muscles and convulsions. Also incontinence of feces and urine is common. Death frequently occurs and is of the asphyxial type.

Nervous symptoms are commonly found. In these

cases the knee jerks were as a rule almost always absent during the acute stage. Marked mental symptoms often remained for weeks; and irritability, confusion and delusions are not uncommon. In reference to the eyes nystagmus and squint with diplopia was frequently seen (66).

Multiple neuritis associated with weakness and marked wasting of the legs was found, and the tibialis anticus muscle was the muscle most frequently attacked. Lumbar puncture on most cases revealed a cerebro-spinal fluid which was clear and sterile with a pressure which was above normal (66).

Gradwahl & Schisler (31) thought that the main pathology was a retention problem; and such metabolic end-products as urea, nitrogen, creatinin and uric acid had a paralyzing action on the nervous tissue. Thus the degree of autointoxication in a measure influenced the nervous symptoms resulting. Their contention was that heat stroke was unquestionably analogous to the symptomatology in some of the forms of uremia.

PATHOLOGY

At the autopsy table in 1863 Wood (69) found that

the blood was very fluid, dark in color and had an acid reaction and that it seemed to have lost its power of coagulating. The heart was rigidly contracted and the veins were markedly congested with blood.

Smith (59) made the following observations:

1. Circulatory Findings:

The arterioles were contracted and the blood volume less. The venous system was engorged and there were small hemorrhages beneath the endocardium and pericardium.

2. Pulmonary Findings:

There seemed to be present an acute intoxication with edema and infiltration. There was interstitial infiltration in the alveolar walls massive enough to be almost complete consolidation. The Bronchi were constricted and the mucous was thrown into folds.

3. Gastro-Intestinal Findings:

The colon reveals marked constriction and some congestion. The liver shows marked drainage and degeneration. The pancreas shows marked parenchymatous degeneration. The spleen is hemorrhagic. The kidneys show extensive damage

in the epithelium of the convoluted tubules.

The brain shows advanced degree of parenchymatous degeneration.

McKenzie and LeCount in working on the nervous systems observed the following changes: There are minute hemorrhages throughout the brain and leptomeninges. They also found varying amounts of cells in the edema of the leptomeninges which has lead to the view that there may be a serious meningitis in heatstroke. There is usually edema of the brain or leptomeninges or of both. There is usually a marked generalized passive hyperemia especially the brain tissue (46).

Some of the residual nervous symptoms (Weisenburg-65) noted following heat stroke are quite numerous and varied. There has been seen acute cerebellor ataxia, loss of speech and spastic symptoms, mental changes, inability to stand heat, and motor lesions, either hemiplegic or paraplegic. Sensory changes have not occurred, however, in any of the cases. Also there has been mention of tremors, different forms of psychoses, facial palsy, neuroses and hysteria (65).

The severity of the heat stroke by no means determines the amount or extent of the cerebral symptoms.

In fact the amount of cerebral or nervous symptoms seems to be dependant upon the individual susceptibility which cannot be measured or standardized in any way.

In reviewing briefly the above symptoms and pathological changes, it can readily be seen that heat-stroke doesn't in any measure seem to be limited in its effect on the body. Rather it has the ability to affect any part or most of the body.

TREATMENT

In heat stroke immediate steps must be taken to reduce the temperature until it reaches 102°F. There have been methods advanced but all are some application of hydrotherapy. Some of the more common forms used are:

1. Cold towel packs.

Large turkish towels are wrung out in water at 60°-70°F. These are applied to the arms, trunk and legs of the patient and brisk massage done. As soon as the towels get warm, fresh cool towels are applied. This is kept up continually until the temperature drops the desired amount then a brisk alcohol rub is given and the patient wrapped in

blankets. When the temperature again begins to go up this is repeated.

2. Cold tubs.

The patient is placed in a large tub in which pieces of ice are floating. Then constant rubbing is done all the while the patient is in the tub. The rectal temperature is taken every five minutes, and when it has dropped to about 102°F. the patient is removed and placed in a warm bed. As soon as the temperature rises the process is repeated.

3. Cold Friction:

In this procedure the patient is rubbed vigorously with large pieces of ice until the temperature is reduced.

4. Cold Water:

Cold water is dashed on the patient or poured on the patient from great heights until a reduction of temperature has been accomplished.

5. Cold or Ice Water Enemas:

Frequent with any of the above procedures cold or ice water enemas are given as supplementary treatment.

As these cases frequently have convulsions they can often be controlled by small whiffs of chloroform,

opiates used advisedly, venesection of ten to twenty ounces of blood, and the barbiturate compounds. A very effective one being sodium amytal grains $3\frac{1}{2}$ -7 intravenously. The cardiac failures should be taken care of by the use of digitalis, strychnine, adrenalin, and the pituitary extracts. The respiratory failures were treated by means of artificial respiration, CO₂ and oxygen, or oxygen bubbled through alcohol.

As cerebral edema is one of the frequent complications it can effectively be treated by:

1. Lumbar puncture and letting off small amounts of cerebrospinal fluid.
2. Intravenous glucose 50-120 c.c. of 50%, or Succrose 100-250 c.c. of 50% (66, 37, 40, 39, 43, 46, 54, 63, 68).

However as the symptoms and pathology of heatstroke vary so greatly, the treatment in many respects largely depends upon the facilities at hand and the viewpoint of the attending physician.

INCIDENCE AND MORBITY

Heatstroke is more frequently found in those parts of the country that have a relatively high

humidity and periods of rather long sustained high temperatures.

It is found more frequently in men than in women, and more often in the laboring man.

The age incident is high in the first year of life and then it drops off sharply until about the age of twenty when it begins to rise slowly. After the age of seventy the incidences again raise sharply.

It is responsible for 0.91 deaths in every 100,000 males and 0.37 deaths in every 100,000 females.

Those states having a high colored population have a death rate 2-4 times as high for the colored as for the white population. Deaths are reported from excessive heat in every month of the year indicating industrial conditions as causative of many cases (55, 27, 8).

SUMMARY

Briefly I have attempted to present here, not a comprehensive survey of any one of the heat manifestations, nor all of them, but rather to broadly cover the field. Thus in this broad survey to give some conception of the types, the mechanism of production,

the pathology produced, the treatment involved and the incidence of the various heat manifestations.

Heat exhaustion is a disorder which is characterized by faintness, weakness, dizziness, and shock-like conditions. Usually having a normal to subnormal temperature and having little or no residual effects. Heat cramps on the other hand is a manifestation characterized by cramps in various parts of the skeletal and smooth muscles. It is associated with nausea and vomiting, and marked disturbance of salt-water metabolism. It likewise has little residual effects.

Heatstroke, however, is the most serious. It is marked by sudden extreme rises in temperature, cardiac and respiratory failures, and mental and motor disturbances. It is often fatal and if not fatal usually leaves permanent motor and mental changes.

CONCLUSIONS

Heat manifestations and hyperpyrexial conditions are often overlooked, and too frequently not considered as very important.

The conditions especially in industry have vital importance from the economical standpoint alone,

because many of the labor hours that are lost are due to heat alone.

The disease in many instances is entirely preventable by treatment that is in its initial cost far less expensive than that expended in hospitalization, loss of working hours and loss in life.

The manifestations are not seasonal, but occur every month during the year.

Thus because of these factors alone I feel that it warrants more attention and study than has been previously given it.

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